Compressive Myelopathy Mimicking Transverse Myelitis

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Objectives: Spinal cord compression may be associated with a fusiform cord lesion on T2-weighted magnetic resonance imaging (MRI) images, leading to confusion with transverse myelitis and delaying effective surgical treatment.

Results: We describe 5 patients referred for evaluation of suspected neuromyelitis optica in whom the final diagnosis was symptomatic cervical spinal stenosis. The patients had gradually progressive myelopathy, with symptoms progressing over an average of 34.4 weeks. Although cervical spinal cord MRI identified long T2 hyperintense lesions, gadolinium enhancement was localized to the level of maximum spinal cord compression, in contrast to the much more extensive distribution of gadolinium enhancement characteristic of myelitis. Compressive myelopathy symptoms responded poorly to corticosteroids, but responded well to surgical decompression.

Conclusion: Cervical cord compression due to spinal stenosis may lead to long intramedullary fusiform T2 hyperintensity on MRI, mimicking inflammatory myelopathy, but the diagnoses can be accurately distinguished by a combination of clinical and radiologic characteristics.

Key Words: spinal cord compression, myelopathy, transverse myelitis, Devic's syndrome, neuromyelitis optica

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Compressive myelopathy is occasionally associated with fusiform T2 signal changes on magnetic resonance imaging (MRI).[1–3] When this occurs, the radiologic appearance can result in misdiagnosis as transverse myelitis, either idiopathic or as a neuromyelitis optica (NMO) spectrum disorder.[4] This misdiagnosis can delay appropriate surgical referral, and may result in ineffective or inappropriate antiinflammatory or immunosuppressive treatments. Our clinical experience in 5 such cases has revealed clinical and radiologic findings that greatly facilitate the recognition of this highly treatable surgical condition and its differentiation from transverse myelitis.

METHODS AND RESULTS

Five individuals were referred for suspected NMO spectrum disorder,[4] due to long spinal cord lesions identified on spinal cord MRI. Ultimately, we determined that these lesions were caused by compressive myelopathy. We reviewed all clinical, demographic, and radiologic data. A neuroradiologist (B.J.E.) characterized the neuroimaging findings.

Illustrative Case Description

A 42-year-old man developed tingling and decreased sensation in the fingers of his right hand. Over the next 2 months, sensory disturbance gradually extended to involve his right hemibody and he developed right leg weakness and gait unsteadiness. MRI of the cervical spine (compressive case 1 in Fig. 1) demonstrated T2 hyperintensity from C5–C7. There was focal intraparenchymal contrast enhancement adjacent to a bulging C6 disk. MRI of the brain and cerebrospinal fluid analysis were normal. He received intravenous followed by maintenance oral corticosteroids with a presumptive diagnosis of transverse myelitis. Neither this treatment nor intravenous immunoglobulin treatment resulted in clinical or radiographic improvement. After 13 months of progressive neurologic symptoms, reevaluation changed the diagnosis to compressive myelopathy because of cervical spondylosis and he was referred for surgical intervention. Following a C4–C7 laminectomy, his symptoms stabilized and subsequently improved over the 6 months after surgery, although there was a gradual and slow improvement in the radiologic findings, with decreasing but persisting enhancement for up to 1 year.

Case Series

The mean age of symptom onset was 51 ± 6.7 years. Four of 5 patients (80%) were men. The average duration of symptomatic progression was 34.4 ± 18.7 weeks. All 5 were treated one or more times with intravenous corticosteroids, but none experienced improvement. No patient had an episode suggesting optic neuritis, and none had prior or subsequent episodes of recurrent myelopathy. The average follow-up was 27.4 ± 20.7 months.

Spinal cord MRI identified T2 hyperintensity corresponding to 3.2 ± 1.6 vertebral levels; this was fusiform and homogenous in 4 (80%). Focal enhancement (defined as less than the height of one vertebral body) following gadolinium administration was identified in all 5 patients, and in all 5, this occurred at the point of maximal focal stenosis. Serum NMO IgG testing was negative in all 5 patients.

Focal enhancement following gadolinium administration was identified in all 5 patients.

Table 1 summarizes data regarding these 5 patients.

DISCUSSION

Patients with longitudinally extensive high T2 signal abnormality on spinal cord imaging in the setting of clinical transverse myelitis are commonly suspected of having a limited or inaugural presentation of NMO ("NMO spectrum disorder"). Forty percent of patients with a first longitudinally extensive transverse myelitis, defined as having an MRI lesion within the cord extending over 3 or more vertebral segments in the setting of an acute myelopathy, are NMO-IgG seropositive and at risk for further attacks of transverse myelitis.[5] However, other causes

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for long spinal cord lesions in the setting of myelopathy, such as symptomatic spinal stenosis, are important to recognize.

Forty percent of patients with a first longitudinally extensive transverse myelitis are neuromyelitis optica-IgG seropositive.

All 5 of our patients with compressive myelopathy experienced progressive neurologic dysfunction over months, as reflected in the mean duration from onset to symptom nadir or intervention, whereas patients with transverse myelitis characteristically reach a nadir of deficit within 2 weeks. None of these 5 patients experienced recurrent myelopathy or optic neuritis, with a mean follow-up duration of 27.4 months. NMO spectrum disorders are characterized by recurrent episodes of myelopathy or optic neuritis.

In all 5 compressive myelopathy cases, gadolinium enhancement was limited to the region of maximal spinal cord compression.

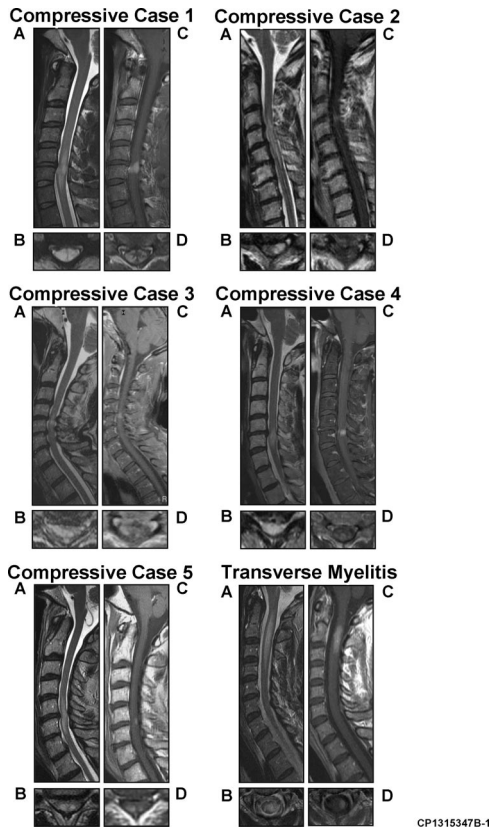

FIGURE 1. Representative sagittal (A) and axial (B) T2 weighted images and sagittal (C) and axial (D) postcontrast images. The cases of compressive myelopathy are labeled compressive cases 1 to 5. In all 5 cases, focal gadolinium enhancement occurs at the level of maximum stenosis. A representative case of transverse myelitis shows a long segment of contrast enhancement and the absence of central canal stenosis.

TABLE 1. Demographic, Clinical and Radiographic Data

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Demographics					
Onset Age (yr)	42	48	60	51	54
Sex	M	M	F	M	M
Clinical					
Weeks to symptom nadir or intervention	16	40	60	40	16
Months follow-up	11	27	41	54	4
MRI					
T2 lesion extent (no. levels)	2	5	5	2	2
Cord enlargement	Y	N	N	Y	N
Serology					
NMO IgG positive	N	N	N	N	N

F indicates female; M, male; N, no; Y, yes.

In inflammatory myelopathy, enhancement characteristically occurs over a longer segment of the cord. Similar to the findings in cases of idiopathic transverse myelitis or NMO-related myelitis, longitudinally extensive regions of T2 hyperintensity, greater than 2 vertebral segments, were present. The pathology of extensive T2 hyperintensity in compressive myelopathy, and its relationship to outcome from surgical or conservative management, remains uncertain. Proposed pathologic substrates of this T2 signal abnormality have included myelomalacia, gliosis, shear-stress injury, edema related to vascular compromise or inflammation, inflammatory demyelination, and vacuolar changes.[1,6–8] Conflicting reports relating T2 signal changes to treatment outcome further complicate the issue. The general trend toward neurologic recovery among our patients, even after a prolonged presurgical course, demonstrates that pathology is partially reversible, consistent with findings of others that increased spinal cord intensity is either not associated or weakly associated with outcome.[1,9] One explanation for the progressive worsening of myelopathy before surgery is reversible inflammation or edema which contributes to cord enlargement, T2 signal abnormality and gadolinium enhancement in the region of stenosis.

Some authors have proposed that central nervous system trauma incites inflammatory demyelinating lesions in patients with preexisting multiple sclerosis; one author has cited the example of cord lesions occurring at the sites of spondylosis and stenosis.[10] Others present contrary arguments.[11] None of the compressive cases in our series improved or stabilized following intravenous corticosteroids, in contrast to the clinical experience with inflammatory myelopathies, where many patients improve after corticosteroid administration and some improve spontaneously, arguing against an inflammatory demyelinating etiology induced by trauma. All 5 patients in the compressive myelopathy group improved or stabilized after surgical intervention. None of these patients had previous or subsequent events suggestive of demyelination. These observations are consistent with the assertion that the patients had reversible cord edema or ischemia related to compression rather than inflammatory demyelination and support the position that clinical and radiographic findings may distinguish those patients who would benefit from surgical decompression.[12]

CONCLUSIONS

Cervical spinal cord compression may be associated with longitudinally extensive fusiform regions of T2-weighted intramedullary

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signal hyperintensity on MRI that may suggest inflammatory or neoplastic myelopathies. However, the combination of clinical and radiologic features can distinguish these cases from inflammatory myelopathies, and surgical referral is recommended when appropriate.

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